

Childhood Sexual Abuse and Bulimic Behavior in a Nationally Representative Sample

ABSTRACT

Objectives. This study examines whether childhood sexual abuse is a significant risk factor for the development of bulimic behavior in women.

Methods. Interviews were conducted among a representative national sample of 1099 US women regarding their sexual experiences during childhood as well as their lifetime prevalence of bulimic behavior. Logistic regression was used to calculate adjusted odds ratios to measure the contribution of childhood sexual abuse to the odds of several measures of bulimic behavior in the population, controlling for the respondent's age, ethnic group, and parents' educational level. Attributable risk analyses were conducted to estimate how much risk for bulimic behavior was due to childhood sexual abuse.

Results. Victims of childhood sexual abuse were significantly more likely to display bulimic behavior, and a substantial fraction of cases of bulimic behavior could be attributed to such abuse.

Conclusions. Childhood sexual abuse is a risk factor for bulimic behavior. Between one sixth and one third of the cases of significant bulimic disturbance could be attributed to childhood sexual abuse. (*Am J Public Health*. 1996;86:1082-1086)

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Introduction

Bulimia nervosa is a serious behavioral disorder that affects approximately 2% of adolescent and young adult females.¹ Its symptoms (recurrent binge eating, a lack of control over eating during such episodes, the purging of food to regulate body weight, and excessive concern about body shape and weight) increase the risk of numerous medical complications and serious social morbidity.² The causes of bulimia nervosa are generally considered to be complex and multifactorial. Frequently identified risk factors include sociocultural pressure for women to be thin, histories of dieting, low self-esteem, body dissatisfaction, and mood instability.³

Recently, there has been an increased interest in the possibility that childhood sexual abuse may also be a risk factor for bulimia nervosa. Retrospective case-control studies have produced contradictory evidence about the relationship between childhood sexual abuse and bulimia nervosa: some studies support the relationship⁴⁻⁶ while others fail to find such an association.⁷⁻⁹ Pope and Hudson¹⁰ reviewed this literature and criticized the studies that reported a significant association between childhood sexual abuse and bulimia nervosa because of problems with the control groups used in each study. These researchers concluded that the findings from six studies with control groups and from numerous studies lacking control groups did not support the hypothesis that childhood sexual abuse was a risk factor for bulimia nervosa.

Such a conclusion may be premature given that some of the studies that found no relationship also had methodological limitations. For example, one study⁸ used

a mixed sample of both anorexic and bulimic individuals and thus did not adequately evaluate whether childhood sexual abuse was related specifically to bulimia nervosa. Another study⁹ that did not find an increased rate of eating disturbance in sexual abuse victims included subjects who were abused as adults; moreover, 68% of the victims were abused on only one occasion, which is not representative of the typical childhood sexual abuse experience, especially that involving intrafamilial abuse.¹¹ Furthermore, all but one of the studies reviewed by Pope and Hudson relied on clinical samples of women who were seeking treatment for eating disorders, which may limit the generalizability of the findings.

Finally, the available studies have relied on research designs and statistics that examine the correlation between childhood sexual abuse and bulimia nervosa, which might be considered a measure of "goodness of fit"¹² between these variables. While such an approach can show how accurately childhood sexual abuse predicts later bulimia nervosa, it cannot show how necessary such abuse may be as a risk factor for the disorder. Other approaches, such as population attributable risk analyses, are needed to determine the magnitude of such a relationship.

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The present study addresses many of these methodological problems by using detailed, structured questions to assess the history of childhood sexual abuse and selected bulimic behaviors in a representative sample of US women in the general population. The general hypothesis for this study is that childhood sexual abuse is a risk factor for bulimic behavior. This hypothesis leads to four predictions about sexually abused women compared with nonabused women: (1) Sexually abused women will be more likely to report binge eating behavior. Clinical theories^{13,14} suggest that psychological traumas such as sexual abuse result in long-term psychobiological deficits in affect regulation that may in turn increase the risk of binge eating as a coping strategy. (2) Sexually abused women will be more likely to express excessive concern about body shape and weight. Clinical studies^{15,16} suggest that victims of childhood abuse often express low self-esteem and body dissatisfaction. (3) Sexually abused women will be more likely to combine both binge eating and purging behaviors (such as vomiting, laxative abuse, and extreme forms of dieting or exercise). If sexual abuse victims are prone to both binge eating and concerns about body shape and weight (as predicted), they should also be more likely to rid themselves of calories through purging to prevent weight gain. (4) Sexually abused women will be more likely to display all three bulimic behaviors under study (binge eating, purging, and excessive weight concern), a set of behaviors that approximate the diagnostic criteria for bulimia nervosa in the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition, revised (DSM-III-R).¹⁷

Methods

Sampling

The data in this study came from a longitudinal survey of drinking behavior in a representative sample of US women living in noninstitutional settings. This national survey was designed to study various behaviors and experiences, including eating disorders and childhood sexual abuse, that might be related to women's drinking. In 1981, female interviewers from the National Opinion Research Center conducted face-to-face interviews with 911 women and 396 men.¹⁸ In 1991, the center reinterviewed 696 of the women from the 1981 sample (age 31 or older), plus a new sample of 403 women aged 21

to 30 (so that the 1991 sample would represent all women 21 or older). (Comparisons of 1981 respondents reinterviewed in 1991 and those not reinterviewed, as well as comparisons of the 1991 sample with US population data, are available from the authors.) Completion rates for women eligible to be interviewed were 81% in 1981 and 87% in 1991. All analyses in the present study use only the current and retrospective data from the 1991 survey. More complete descriptions of the sample are available elsewhere.^{18,19}

Measures

The survey included questions about demographic characteristics; social roles; various forms of psychopathological behavior, including depression, anxiety, bulimic behavior, and substance abuse; and sexual experiences, including childhood sexual abuse. Only data on bulimic behavior, childhood sexual abuse, and demographic characteristics were used in the analyses presented here.

The questions for assessing bulimic behaviors were taken from the Structured Clinical Interview for DSM-III-R (SCID).²⁰ Respondents were asked if they (1) believed they were more concerned about body shape and weight than other people their age; (2) had ever eaten abnormally large amounts of food within a few hours at least twice a week for 3 months or more; and (3) had ever engaged in counteractive behaviors such as vomiting, laxative use, or severe dieting during such eating episodes.

Childhood sexual abuse was measured by detailed questions about sexual experiences before the age of 18. Women were *not* asked if they had been sexually abused; instead, they were asked if they had ever experienced particular sexual acts, including exhibitionism, sexual kissing, touching and fondling of genitals, oral-genital activity, and vaginal and anal intercourse. Based on research by Wyatt,²¹ childhood sexual abuse was defined as (1) any intrafamilial sexual activity before age 18 that was unwanted by the respondent or that involved a family member 5 or more years older than the respondent; and (2) any extrafamilial sexual activity that occurred before age 18 and was unwanted, or that occurred before age 13 and involved another person 5 or more years older than the respondent. Voluntary experiences between ages 13 and 17 with partners 5 or more years older were not included in the definition of abuse.

Analyses

Responses were weighted to compensate for stratified sampling (based on age and drinking levels) and for nonresponse rates in subdivisions of the sample. Because cluster sampling may have modified variances of respondent characteristics (design effects), responses were further weighted to reduce sample sizes for significance testing to two thirds of the number of respondents actually interviewed. Details of the weighting procedures are available on request.

Adjusted odds ratios (with 95% confidence intervals) were calculated with logistic regression to estimate how childhood sexual abuse affected the odds of each measure of bulimic behavior. Calculations were made after controlling for the effect of respondent's age (dichotomizing age at 47 years, which maximizes the strength of the relationship of age with childhood sexual abuse and bulimic behavior and is identified as the appropriate cut point by the MAMBAC procedure²²), ethnic category (White non-Hispanic vs all others), and parents' educational level (for the parent with the most education: 8th grade or less, 9th grade through high school, or more than high school). A likelihood ratio chi-square goodness-of-fit test of a no-interaction logit model found no significant interaction.²³

The odds ratios were used to calculate population attributable risk statistics to estimate how much the risks for bulimic behavior depended on childhood sexual abuse. Population attributable risk indicates the percentage of cases (i.e., of bulimic behavior) that can be directly attributed to the experience of a particular risk factor (i.e., childhood sexual abuse). This statistic has been used to evaluate risk factors for premature birth,²⁴ hypertension,²⁵ and lung cancer,²⁶ and to examine the impact of stressful life events on depression.¹² Scott²⁷ used attributable risk analyses to show the impact of childhood sexual abuse on risks of eight different psychiatric disorders, but bulimia nervosa was not assessed in that study.

The population attributable risk, adjusted for the control variables, was estimated as

$$AR_c = (x - y)/x,$$

where x is the (weighted) number of bulimic cases in the sample and y is an estimate of the number of bulimic cases that would have occurred if no one in the sample had had a history of childhood

TABLE 1—Odds Ratios and Measures of Population Attributable Risk for Bulimic Behaviors in a Nationally Representative Sample of US Women

Bulimic Behaviors	Abused ^a (n = 157), No. (%)	Not Abused ^a (n = 497), No. (%)	Odds Ratio ^b	95% CI	Population Attributable Risk
Binge eating	30 (19.1)	39 (7.8)	1.96 ^c	1.13, 3.40	.17
Weight concern	58 (37.1)	153 (34.7)	1.41	0.94, 2.12	.05
Binge + weight concern	17 (10.6)	24 (4.9)	1.83	0.91, 3.68	.16
Binge + counteract	12 (7.7)	10 (2.0)	2.62 ^c	1.05, 6.57	.30
Binge + counteract + weight concern	8 (5.4)	6 (1.2)	3.17	0.99, 10.13	.34

^aAlthough 1099 respondents were interviewed, the weighted n for statistical analyses was reduced to 733 to compensate for design effects and was further reduced to 654 because of missing data.

^bAfter controlling for age, ethnicity, and parental education.

^cSignificant at the .05 level.

sexual abuse. The value of y is based on logistic regression of bulimia on childhood sexual abuse and the control variables

$$y = \sum_j n_j \hat{f}_j^*,$$

where the summation is over all cells j of the cross-classification of childhood sexual abuse and the control variables; n_j is the weighted number of individuals (bulimic cases and controls) in cell j ; and \hat{f}_j^* is the probability that an individual characterized by the values of the control variables in cell j but without childhood sexual abuse is bulimic, as estimated by logistic regression.²⁸ This statistic, when multiplied by 100, can be interpreted as the estimated percentage of bulimic cases in the population that would not have occurred without childhood sexual abuse.

Results

Twenty-four percent of the women surveyed were classified as having been sexually abused in childhood. (Additional details about the estimation of the prevalence of such abuse are available from the authors.) This estimate is consistent with estimates of the prevalence rates for childhood sexual abuse for women in the general population.²⁹ Table 1 shows the percentages of sexually abused and non-abused women in the 1991 sample who reported various behaviors associated with bulimia nervosa. Binge eating, by itself or in combination with counteractive behaviors, was two or more times more prevalent among the women who had experienced childhood sexual abuse than among the women who had not. In contrast, heightened concerns about weight and body shape, although much more prevalent than bingeing and purging, were not

related as strongly to childhood sexual abuse.

Adjusted odds ratios, derived from logistic regressions that took into account effects of age, ethnicity, and parental education, confirm what the percentages suggest. Childhood sexual abuse significantly increased the likelihood that women would engage in binge eating, by itself or in combination with counteractive behaviors. However, such abuse was much less relevant to women's odds of being highly concerned about their weight and body shape.

Attributable risk analysis was used to estimate what percentage of cases of bulimic behavior would not have occurred if women had not been sexually abused as children. The analyses suggest that as many as 17% of women reporting binge eating experiences, and 30% of women reporting a history of binge eating combined with counteractive measures, would not have had these bulimic experiences in the absence of childhood sexual abuse. The statistics also indicate that the combination of bingeing and counteractive behaviors with intensified weight concerns was contingent on childhood sexual abuse in 34% of the cases. In short, childhood sexual abuse was apparently an essential precondition for between one sixth and one third of the more serious cases of bulimic behavior among women in the general population.

Discussion

The analyses of prevalence rates, odds ratios, and attributable risks lead to two conclusions: (1) experiences of childhood sexual abuse by themselves are unlikely to be sufficient to cause bulimic behavior, but (2) a large fraction of

women who engage in binge eating and purging would not do so if they had not been sexually abused as children. These conclusions are consistent with the general hypothesis that childhood sexual abuse is a risk factor for bulimic behavior. The conclusions are strengthened by use of a representative national sample of women in the general population, multivariate analyses, and rigorous criteria for identifying childhood sexual abuse. Two other surveys that relied on representative national samples of women (in the United States and Canada) have also recently revealed a similar association between childhood sexual abuse and bulimia nervosa.^{30,31}

The prediction that victims of childhood sexual abuse would be more concerned about body shape and weight than nonvictims was not supported. The high prevalence of weight concerns among the women interviewed (more than 30%) suggests that weight preoccupation may be so common among US women today that it has many causes unrelated to childhood sexual abuse. Failure to find a statistically significant relationship between childhood sexual abuse and excessive weight concern suggests that the abuse may be more closely linked to overt behavioral aspects of bulimia nervosa (i.e., bingeing and purging) than to cognitive aspects of the disorder (i.e., excessive weight concern). This would be consistent with the idea that bingeing and purging behaviors of childhood sexual abuse victims are instrumental efforts to cope with negative affect associated with their abuse.¹³ Childhood sexual abuse may be a traumatic experience that results in long-term affective dysregulation and in bodily shame,³² and that increases the risks of overt coping behaviors such as alcohol consumption,^{33–35} self-destructive behavior,^{36,37} and bingeing and purging. This pattern would be consistent with a recent clinical case-control study indicating that sexual abuse is a nonspecific risk factor for bulimia nervosa as well as for other psychiatric disorders.³⁸

The results presented here suggest that more than one fourth of the women in the United States who have engaged in a combination of binge eating and counteractive behaviors would not have behaved this way had they not been sexually abused as children. However, because only a minority of such victims report these behaviors, it is difficult to predict which abuse victims will develop bulimic behavior. It is similarly difficult to predict cases of lung cancer, where there is a low

correlation between smoking and lung cancer³⁹ but as many as 85% of the cases can be attributed to cigarette smoking.²⁶ This problem arises because while very few nonsmokers develop lung cancer, many smokers do not develop the disease either. Similarly, risk factors other than childhood sexual abuse (e.g., body image concerns, extreme dieting, mood disturbances, social relationship problems) must be considered to determine which abuse victims will develop bulimia nervosa. Consistent with the general belief that the cause of bulimia nervosa is multifactorial,^{2,3} the results here indicate that childhood sexual abuse should be included as a risk factor in multifactorial models of bulimia nervosa.

The survey used here did not ask specifically about a lack of control during binge eating, one of the DSM-III-R criteria for bulimia nervosa. However, the 2.2% of respondents who reported all three measured bulimic behaviors is close to the estimated population prevalence of DSM-III-R bulimia nervosa, which ranges from 1.1% to 1.7% in community-based samples.^{31,40} The survey also did not verify that the onset of bulimic behavior occurred later than childhood sexual abuse. However, restricting the measurement of sexual abuse to experiences before age 18 makes it likely that the abuse occurred before the typical age of onset for bulimia nervosa. Other studies in which the sequences of childhood sexual abuse and bulimia nervosa could not be verified have made similar assumptions.¹⁰

Pope and Hudson¹⁰ raise four methodological issues that need to be considered in evaluating any study that examines the relationship between childhood sexual abuse and bulimia nervosa. First, studies comparing the prevalence rates of childhood sexual abuse in samples of individuals seeking treatment for bulimia nervosa with such rates in the general population may be biased. Repeated therapeutic interviews in clinical studies may elicit higher rates of abuse than does the single-interview approach that is typically used in general population studies. This issue is not relevant to the present study because all subjects received the same single interview.

Second, if interviewers are aware of the subjects' bulimic behavior and suspect a link between childhood sexual abuse and bulimia, they may elicit responses that confirm their suspicions. In the present study, the interviewers asking about childhood sexual abuse knew the subjects' responses about bulimic behav-

ior but were unaware of any predictions about the relationship between these variables. Furthermore, the interviewers asked about many adverse experiences and psychopathological conditions, making it unlikely that they made any specific inference about the relationship between bulimic behavior and childhood sexual abuse.

Third, the relationship between these two variables may be inflated if bulimic patients, in an "effort after meaning," search their lives for an explanation for their bulimia nervosa and consequently remember or report childhood sexual abuse more often than do control subjects, who lack such explanatory motivation. The present study cannot rule out the possibility that some reports of childhood sexual abuse were influenced by the effects of such a search for meaning. However, none of the respondents with bulimic behavior (i.e., those displaying bingeing, counteracting behavior, and excessive weight concern) in this study endorsed an item in the interview indicating that they had only a "vague" memory of childhood sexual abuse. Instead, their accounts of their sexual experiences contained more information than would be needed for effort after meaning. Also, questions about sexual experiences were separated from questions about bulimic behavior by other topics (e.g., marital relationships, gambling, and adult sexual experience), thus reducing any demand characteristic that may have increased the likelihood of an interview-based association of childhood sexual abuse and bulimic behavior. Furthermore, as noted previously, there was no reference to sexual abuse in the interview; respondents were asked only about sexual experiences. Consequently, there is nothing in the content or position of the interview questions that would encourage effort after meaning.

Fourth, Pope and Hudson suggest that a family history of psychiatric problems may increase risks of both childhood sexual abuse and bulimia nervosa, thereby magnifying their apparent relationship. To show that such abuse contributes to risks for bulimia nervosa, they suggest that one must demonstrate a significantly higher prevalence of childhood sexual abuse in bulimic samples after controlling for effects of family psychiatric disturbances, such as alcoholism and depression. Although such analyses might help to clarify the network of multiple influences on bulimia nervosa, they are beyond the scope of this study. However, the logic

of the recommended analyses implies that only independent main effects are important for understanding what causes bulimia nervosa. Childhood sexual abuse may mediate or interact with effects of family psychiatric history on risks for bulimia. Causal models that emphasize a single cause or that assume an unwarranted incompatibility between alternative causal models may have limited predictive ability and may oversimplify clinical decision making.⁴¹ What is needed is to integrate childhood sexual abuse with other risk factors, including family psychiatric history, in multivariate causal models of bulimia nervosa. □

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References

1. American Psychiatric Association. Practice guidelines for eating disorders. *Am J Psychiatry*. 1993;150:207-228.
2. Mitchell JE. *Bulimia Nervosa*. Minneapolis, Minn: University of Minnesota Press; 1990.
3. Johnson C, Connors ME. *The Etiology and Treatment of Bulimia Nervosa*. New York, NY: Basic Books; 1987.
4. Steiger H, Zanko M. Sexual traumata among eating disordered, psychiatric and normal female groups. *J Interpersonal Violence*. 1990;5:74-86.
5. Hall RCW, Tice L, Beresford TP, Wooley B, Hall AK. Sexual abuse in patients with anorexia nervosa and bulimia. *Psychosomatics*. 1989;30:73-79.
6. Miller DAF, McCluskey-Fawcett K. The relationship between childhood sexual abuse and subsequent onset of bulimia nervosa. *Child Abuse Negl*. 1993;17:305-314.
7. Folsom V, Krahn D, Nairn K, Gold L, Demitrack MA, Silk VR. The impact of sexual and physical abuse on eating disordered and psychiatric symptoms: a comparison of eating disordered and psychiatric inpatients. *Int J Eating Disord*. 1993;13:249-257.
8. Ross CA, Heber S, Norton GR, Anderson G. Differences between multiple personality disorder and other diagnostic groups on structured interview. *J Nerv Ment Dis*. 1989;21:169-189.
9. Finn SE, Hartman M, Leon GR, Lawson L. Eating disorders and sexual abuse: lack of confirmation for a clinical hypothesis. *Int J Eating Disord*. 1986;5:1051-1060.
10. Pope HG, Hudson JI. Is childhood sexual abuse a risk factor for bulimia nervosa? *Am J Psychiatry*. 1992;149:455-463.

11. Finkelhor D. The sexual abuse of children: current research reviewed. *Psychiatr Ann*. 1987;17:233-241.
12. Cooke DJ, Holes DJ. The aetiological importance of stressful events. *Br J Psychiatry*. 1983;143:397-400.
13. Briere J. *Child Abuse Trauma: Theory and Treatment of Lasting Effects*. Newbury Park, Calif: Sage Publications; 1992.
14. Van der Kolk BA. *Psychological Trauma*. Washington, DC: American Psychiatric Press; 1987.
15. German DE, Habenicht DJ, Fitcher WG. Psychological profile of the female adolescent incest victim. *Child Abuse Negl*. 1990;14:429-438.
16. Van der Velde CD. Body images of one's self and of others: developmental and clinical significance. *Am J Psychiatry*. 1985;142:527-537.
17. American Psychiatric Association. *The Diagnostic and Statistical Manual for Mental Disorders*. 3rd ed, rev. Washington, DC: American Psychiatric Press; 1987.
18. Wilsnack RW, Wilsnack SC, Klassen AD. Women's drinking and drinking problems: patterns from a 1981 national survey. *Am J Public Health*. 1984;74:1231-1238.
19. Wilsnack SC, Wilsnack RW. Drinking and problem drinking in US women: patterns and recent trends. In: Galanter M, ed. *Recent Developments in Alcoholism. Volume 12. Alcoholism and Women*. New York, NY: Plenum; 1995.
20. Spitzer RL, Williams JBW, Gibbon M, First MB. *The Structured Clinical Interview for DSM-III-R*. New York, NY: New York State Psychiatric Institute; 1988.
21. Wyatt G. The sexual abuse of Afro-American and White American women in childhood. *Child Abuse Negl*. 1985;9:507-519.
22. Meehl PE, Yonce LJ. Taxometric analysis, I: detecting taxonicity with two quantitative indicators using mean above and below a sliding cut (MAMBAC procedure). *Psychol Rep*. 1994;74:1059-1274.
23. Agresti A. *Categorical Data Analysis*. New York, NY: John Wiley & Sons; 1990.
24. Feldman JG, Minkoff HL, McCalla S, Salwen M. A cohort study of the impact of perinatal drug use on prematurity in an inner-city population. *Am J Public Health*. 1992;82:726-728.
25. Larbi ED, Stamler J, Dyer A, et al. The population attributable risk of hypertension from heavy alcohol consumption. *Public Health Rep*. 1984;99:316-319.
26. Chyou P-H, Nomura AMY, Stemmermann GN. A prospective study of the attributable risk of cancer due to cigarette smoking. *Am J Public Health*. 1992;82:37-40.
27. Scott KD. Childhood sexual abuse: impact on a community's mental health status. *Child Abuse Negl*. 1992;16:285-295.
28. Bruzzi P, Green SB, Byar DP, Brinton LA, Schairen C. Estimating the population attributable risk for multiple risk factors using case-control data. *Am J Epidemiol*. 1985;122:904-914.
29. Finkelhor D. Current information on the scope and nature of child sexual abuse. *Sex Abuse Child*. 1994;4:31-53.
30. Dansky BS, Brewerton TD, Kilpatrick DG, O'Neil PM. The national women's study: relationship of victimization and PTSD to bulimia nervosa. *Int J Eating Disord*. In press.
31. Garfinkel PE, Lin E, Goering P, et al. Bulimia nervosa in a Canadian community sample: prevalence and comparison of subgroups. *Am J Psychiatry*. 1995;152:1052-1058.
32. Andrews B. Bodily shame as a mediator between abusive experiences and depression. *J Abnorm Psychol*. 1995;104:277-285.
33. Miller BA, Downs WR, Gondoli DM, Keil A. The role of childhood sexual abuse in the development of alcoholism in women. *Violence Victims*. 1987;2:157-172.
34. Rohsenow DJ, Corbett R, Devine D. Molested as children: a hidden contribution to substance abuse? *J Subst Abuse Treat*. 1988;5:13-18.
35. Russell SA, Wilsnack SC. Adult survivors of childhood sexual abuse: substance abuse and other consequences. In: Roth P, ed. *Alcohol and Drugs Are Women's Issues*. Vol. 1. *A Review of the Issues*. New York, NY: Women's Action Alliance; 1991.
36. Van der Kolk BA. The compulsion to repeat the trauma: re-enactment revictimization and masochism. *Psychiatr Clin North Am*. 1989;12:389-411.
37. Van der Kolk BA, Perry JC, Herman JL. Childhood origins of self-destructive behavior. *Am J Psychiatry*. 1991;148:1665-1671.
38. Welch SL, Fairburn CG. Sexual abuse and bulimia nervosa: three integrated case control comparisons. *Am J Psychiatry*. 1994;151:402-407.
39. Doll R, Peto R. Mortality in relation to smoking: 20 years' observations on male British doctors. *BMJ*. 1976;2:1525-1536.
40. Crowther JH, Wolf EM, Sherwood NE. Epidemiology in bulimia nervosa. In: Crowther JH, Tennenbaum DL, Hobfall SE, Stephens MAP, eds. *The Etiology of Bulimia Nervosa: The Individual and Familial Context*. Washington, DC: Hemisphere Publications; 1992.
41. Haynes SN. *Models of Causality in Psychopathology: Toward Dynamic, Synthetic Nonlinear Models of Behavior Disorders*. New York, NY: MacMillan Publishing; 1992.